Journal of Arrhythmia 30 (2014) 466–472

Contents lists available at ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Original Article

Changes over time in echocardiographic variables and atrial electromechanical intervals after ablation for atrial fibrillation

Hiroaki Mano, M.D., Yasuo Okumura, M.D.*, Ichiro Watanabe, M.D., Masakatsu Ohta, M.D., Rikitake Kogawa, M.D., Naoko Sasaki, M.D., Toshiko Nakai, M.D., Kimie Ohkubo, M.D., Masayoshi Kofune, M.D., Koichi Nagashima, M.D., Kazumasa Sonoda, M.D., Hironori Haruta, M.D., Atsushi Hirayama, M.D.

Division of Cardiology, Department of Medicine, Nihon University School of Medicine, Tokyo, Japan

ARTICLE INFO

Article history:
Received 11 September 2013
Received in revised form
23 January 2014
Accepted 29 January 2014
Available online 18 March 2014

Keywords:
Atrial fibrillation
Ablation
Total atrial conduction time by tissue Doppler imaging
Reverse remodeling

ABSTRACT

Introduction: Acute and mid-term effects of ablation for atrial fibrillation (AF) on left atrial (LA) and left ventricular (LV) function and the atrial electromechanical interval are controversial.
Methods: Echocardiographic variables and the PA-TDI interval (time from ECG lead II P-wave onset to lateral a′ wave on tissue Doppler tracings, indicating the total atrial conduction time [TACT]) were evaluated in 33 paroxysmal AF patients before, 1 day, and 3, 6, and 12 months after ablation.
Results: During a 12-month follow-up, 10 (30.3%) patients had recurrent AF, associated with a greater baseline LA volume (LAV) (48.4 ± 19.3 mm^3 vs. 38.7 ± 11.4 mm^3, $P=0.0811$) and PA-TDI interval (163.9 ± 11.0 ms vs. 151.1 ± 14.6 ms, $P=0.0189$) than in patients without AF recurrence. By 6 months after ablation, LAV had decreased progressively in the non-recurrence group (27.9 ± 8.1 mm^3, $P<0.0001$ vs. baseline), but the decrease was modestly significant in the recurrence group (36.3 ± 7.5 cm^3, $P=0.0380$). LV ejection fraction (LVEF) modestly increased 1 day after ablation in both groups and remained unchanged in the non-recurrence group (67.9 ± 8.1% at baseline to 70.5 ± 5.9% at 12 months, $P=0.1711$), whereas it decreased gradually below the baseline value in the recurrence group (68.2 ± 11.3% to 60.9 ± 13.6%, $P=0.1025$). The PA-TDI interval did not change during follow-up in either group, but remained longer in the recurrence group.
Conclusions: The PA-TDI interval may be useful for predicting post-ablation AF recurrences. The patterns of time-course changes in LAV, LVEF, and TACT differ, but the effects of ablation were better in patients without AF recurrence after ablation.

1. Introduction

Over the past 10 years, pulmonary vein isolation (PVI) has been used as an effective therapy for atrial fibrillation (AF) [1]. Nevertheless, the progression of atrial electrical and structural remodeling requires additional aggressive ablation strategies, i.e., a complex fractionated atrial electrogram (CFAE) ablation and/or atrial linear ablation. Despite such treatments, the post-ablation AF recurrence rate is higher in patients treated aggressively for atrial remodeling than in patients with less extensive remodeling of the atria treated with PVI alone [2,3]. Determining the extent of structural and electrical atrial remodeling before ablation is relevant for selecting AF ablation strategies. Structural and electrical atrial remodeling are estimated by the left atrial (LA) size, volume (LAV), a shortened atrial refractory period, or a prolonged total atrial conduction time (TACT) [3–7]. Recently, the time interval in the electrocardiogram (ECG) lead II from onset of the P wave to the lateral mitral annulus a′ velocity wave, as assessed with tissue Doppler imaging (PA-TDI interval), has been shown to represent the electromechanical interval reflecting the TACT [6,8–10]. Therefore, we investigated whether this echocardiographic variable could be used to identify patients prone to AF recurrence following catheter ablation.

There is increasing evidence that the maintenance of sinus rhythm following AF ablation results in reversed structural and electrical remodeling of the atria during follow-up [4,11–15]. This study aimed to elucidate the short- and long-term effects of AF ablation on atrial and left ventricular function and the atrial electromechanical interval as assessed with standard and

* Correspondence to: 30-1 Ohyaguchi-kamicho, Itabashi-ku, Tokyo 173-8610, Japan. Tel.: +81 3 3972 8111; fax: +81 3 3972 1098.
E-mail address: yasuwo128@yahoo.co.jp (Y. Okumura).

http://dx.doi.org/10.1016/j.joa.2014.01.005
1880-4276

tissue Doppler-based echocardiography. Additionally, we sought to analyze the time course of the changes in these variables during the short- and mid-term follow-up periods after AF ablation.

2. Methods

2.1. Study patients

The study group comprised of 33 consecutive patients with paroxysmal AF (spontaneous termination within 7 days; 27 men, 6 women; mean age, 57.5 ± 9.5 years) referred to Nihon University Itabashi Hospital for a first radiofrequency catheter ablation. This study excluded patients with non-paroxysmal AF lasting > 7 days, and those undergoing repeat procedures. All patients provided written informed consent for the electrophysiologic study and ablation procedure. This study was approved by the medical ethics committee of our institution (date of approval: Sep 9th, 2011, approval no. RK-110909-2). Adequate oral anticoagulation was administered for at least 1 month before the procedure. All antiarrhythmic drugs were discontinued for at least 5 half-lives prior to the ablation. Upon hospital admission, a medical history, physical examination, 12-lead ECG, chest radiograph, and transesophageal and transthoracic echocardiograms were obtained. All patients underwent multi-slice computed tomography on a 320-row detector, dynamic volume scanner (Aquilion ONE; Toshiba Medical Systems, Tokyo, Japan) to obtain a 3-dimensional reconstruction of the left atrium and pulmonary veins (PVs).

2.2. Electrophysiologic study and ablation procedure

The procedure was performed under intravenous sedation with propofol and fentanyl as previously described [3]. In brief, after vascular access was obtained, a single transseptal puncture was performed and followed by extensive ipsilateral PVI, guided by double Lasso catheters and by using a 3-dimensional geometric map generated by a NavX (St. Jude Medical, St. Paul, Minnesota) or CARTO (Biosense Webster, Inc., Diamond Bar, California) mapping system [1–3]. A 3.5-mm irrigated-tip catheter (ThermoCool, Navistar, Biosense Webster) was used for ablation. Radiofrequency energy was delivered at a maximum power output of 30 W, and the upper temperature limit was set to 41 °C at a saline irrigation rate of 17 mL/min (CoolFlow Pump; Biosense Webster). The endpoint of PVI was the elimination or dissociation of all PV potentials and the

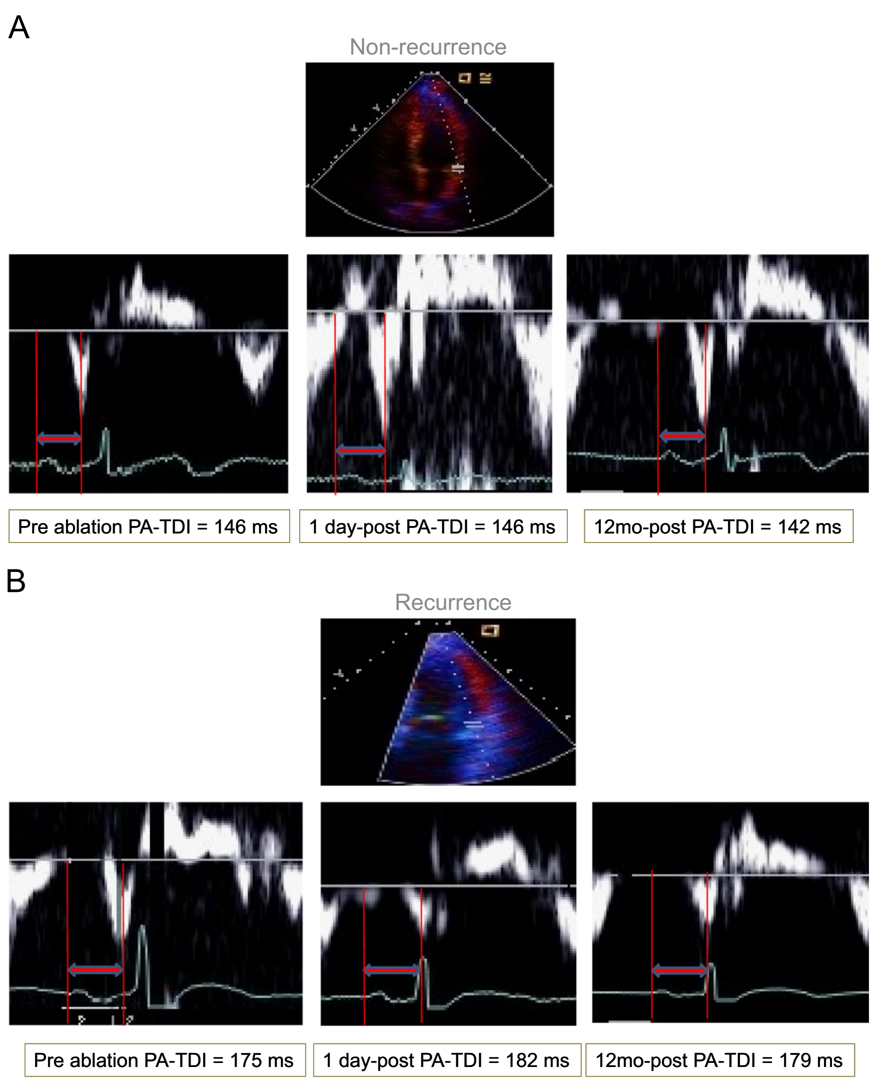

Fig. 1. Examples of the measurement of the PA-TDI interval in patients without (A) and with (B) recurrence of atrial fibrillation after ablation. Upper image shows an apical 4-chamber view and the lower images show tissue Doppler tracings. The PA-TDI is measured as the time interval from the onset of the P wave in ECG lead II to the peak a′ wave of the left lateral atrial wall on the tissue Doppler tracing. The mean PA-TDI intervals in the patient with no AF recurrence are 146 ms before ablation, 146 ms on day 1 after ablation, and 142 ms at 12 months after ablation; in the patient with recurrence, the mean PA-TDI intervals are 175 ms before ablation, 182 ms on day 1 after ablation, and 179 ms at 12 months after ablation.

attainment of complete entrance and exit block. Linear ablations at the left atrial roof and floor along the coronary sinus (CS) and/or CFAE ablations based on the post-PVI CFAE map were performed in patients in whom AF was not terminated by PVI or those in whom sustained AF lasting more than 5 min was inducible by rapid atrial pacing [2,3]. The endpoint of these steps was the termination of AF during the procedure or the abolition of all CFAEs in the left atrium, if AF was not terminated. Cavotricuspid isthmus ablation was also performed when typical atrial flutter could be induced with burst atrial pacing or was observed clinically.

2.3. Echocardiographic evaluation

Transthoracic echocardiography was performed 1 day before ablation and 1 day, 3 months, 6 months, and 12 months after ablation, using an ACUSON Sequoia C256 echocardiography system (Siemens Medical Solutions USA, Inc., Malvern, Pennsylvania). Standardized measurements were obtained: maximum LAV was measured with the prolate-ellipsoid method [16], and left ventricular ejection fraction (LVEF) was assessed with M-mode echocardiography (Teichholz's method). An apical 4-chamber view using Doppler tissue imaging was used to image the mitral annulus, and the following indices of LV diastolic and LA function were measured: (1) the ratio of the mitral inflow early filling velocity (E) to atrial filling velocity (A) (E/A ratio), (2) the ratio of E to the velocity of the septal early mitral annular ascent (e′), (3) the velocity of the a′ wave on the lateral LA tissue Doppler tracing (lateral a′) and (4) the PA-TDI interval (Fig. 1). These variables were measured in 3 consecutive beats and the results averaged. All measurements were performed by two echocardiographers, each with more than 5 years of experience.

2.4. Post-ablation follow-up

The patients' prescribed antiarrhythmic drugs were resumed after the procedure but were stopped after a 2-month post-ablation blanking period. All patients underwent routine follow-up at our outpatient clinic, where a clinical evaluation and 12-lead electrocardiography were performed at 2 weeks and at 1, 3, 6, and 12 months after ablation. We also obtained 24-h Holter recordings at 3, 6, and 12 months after ablation. ECG event monitoring was used whenever the patient reported any cardiac symptoms. Successful ablation was defined as non-recurrence of an AF episode lasting more than 30 s as assessed with a standard ECG, ECG event monitor, or 24-h Holter recording during the 12-month follow-up period after the 2-month post-ablation blanking period. We compared the procedural outcome, baseline characteristics, and transthoracic echocardiography measurements between the patients who had episodes of AF recurrence (AF recurrence group) and non-recurrence (non-recurrence group).

2.5. Statistical analysis

Continuous variables are expressed as mean ± SD or median and interquartile range. Between-group differences were analyzed by using Student's t-test or the Mann–Whitney U test as appropriate. Categorical variables are expressed as percentages, and differences were analyzed with a chi-square test. A paired t test was used to analyze within-group differences in the variables measured at different time points. A P value of <0.05 was considered statistically significant. Statistical analyses were performed with JMP 9 software (SAS Institute, Cary, NC).

3. Results

3.1. Procedural outcome, baseline characteristics, and transthoracic echocardiography measurements in the AF recurrence group and non-recurrence group

In 19 (58%) of the 33 patients, sustained AF could not be induced after PVI. Six (18%) patients required additional LA linear

Table 1
Baseline characteristics, Ablation results, and echocardiographic parameters in patients without and with AF recurrence.

	No recurrence (n=23)	Recurrence (n=10)	P value*
Age, years	57.6 ± 9.9	57.1 ± 9.0	0.8907
Sex: male	20 (87.0)	7 (70.0)	0.2458
AF duration, months	48 (24–73)	57 (16–88)	0.9219
Body mass index, kg/m^2	22.8 ± 2.9	24.7 ± 3.3	0.1152
Casual factors			
Hypertension	8 (34.8)	3 (30.0)	0.7888
Diabetes mellitus	1 (4.4)	2 (20.0)	0.1506
Ischemic heart disease	2 (8.7)	0 (0)	0.3360
Heart failure	0 (0)	1 (10.0)	0.1235
Ablation result			
Terminated by PVI	16 (69.6)	3 (30.0)	0.0346
Terminated by PVI+LA substrate-based ablation	4 (57.1)	2 (28.6)	0.2801
TTE variable			
LAV (cm^3)	38.7 ± 11.4	48.4 ± 19.3	0.0811
LVDd (mm)	48.5 ± 3.9	52.2 ± 6.5	0.0506
LVDs (mm)	30.0 ± 3.9	32.3 ± 8.4	0.2892
LVEF (%)	67.9 ± 68.2	68.2 ± 11.3	0.9493
E/A	1.3 ± 0.5	1.0 ± 0.3	0.1099
DcT (cm/s)	179.5 ± 31.9	179.4 ± 33.4	0.9921
E/e'	6.5 ± 2.0	6.6 ± 1.8	0.9605
Lateral a′ (cm/s)	11.4 ± 4.2	13.6 ± 4.9	0.1909
PA-TDI interval (ms)	151.1 ± 14.6	163.9 ± 11.0	0.0189

AF, atrial fibrillation; DcT, deceleration time; LA, left atrium; LAV, LA volume; LVDd, left ventricular diastolic diameter; LVDs, LV systolic diameter; LVEF, LV ejection fraction; PA-TDI, time interval from onset of the P wave in ECG lead II to the peak a′ wave of the left lateral atrial wall; PVI, pulmonary vein isolation; TTE, transthoracic echocardiography.
Values are the mean ± SD, median (interquartile ranges), or n (%).

* No recurrence vs. recurrence.

ablation and/or CFAE-based ablation to terminate the AF. In the remaining 8 (24%) patients, AF was not terminated despite performance of these ablation steps. During a follow-up period of 12 months, 10 (30.3%) of the 33 patients had AF recurrence. The clinical and echocardiographic characteristics of the AF recurrence group and the non-recurrence group are listed in Table 1. There were no statistically significant differences in the baseline characteristics. As expected, AF that was terminated by PVI was more common in the non-recurrence group than in the recurrence group.

At baseline, LAV and LV diastolic diameter tended to be larger in the AF recurrence group than in the non-recurrence group. The PA-TDI interval was significantly longer in the AF recurrence group than in the non-recurrence group (163.9 ± 11.0 ms vs. 151.1 ± 14.6 ms, $P=0.0189$).

3.2. *Echocardiographic evaluation during follow-up*

Echocardiographic measurements were evaluated in all 33 patients at 3 months (2.8 ± 0.6 months), 6 months (6.3 ± 0.8 months), and 12 months (12.0 ± 1.1 months) after ablation.

3.2.1. *Changes in LA volume and function*

The changes in each of the echocardiographic variables are shown in Table 2. LAV decreased progressively during the 6 months after ablation in the non-recurrence group ($P<0.0001$, baseline vs. 6 months) whereas the decrease was modestly significant in the recurrence group ($P=0.0380$). LAV increased thereafter and returned to approximately baseline levels at 12 months in both groups (Fig. 2A). During the follow-up period, there was no significant improvement in the lateral a′ in either the non-recurrence group or the recurrence group.

3.2.2. *Changes in LV systolic and diastolic function*

In the non-recurrence group, LV diastolic and systolic diameters decreased progressively by 12 months after ablation ($P=0.0187$ and $P=0.0472$, respectively for the values between the baseline vs. 12 months), whereas the decreases were non-significant in the recurrence group ($P=0.0892$ and $P=0.8693$, respectively). In the non-recurrence group, LVEF tended to be increased on day 1 after ablation ($P=0.0905$ vs. baseline value), but thereafter, no further improvement was observed during the follow-up period. In the recurrence group, LVEF tended to be increased 1 day after ablation ($P=0.0783$), but it decreased gradually below the baseline value by 12 months after ablation (Fig. 2B). The E/e' and E/A ratio measured during the follow-up period did not change significantly from the baseline values in either group (Fig. 2C).

3.3. *Impact of the ablation outcome, extensive LA-based ablation strategy, and use of anti-arrhythmic drugs on the PA-TDI interval*

There was no change between the baseline and 12-month post-ablation PA-TDI interval in either the non-recurrence group or AF recurrence group (Fig. 2D). To clarify the effect of the extensive

Table 2
Follow-up transthoracic echocardiographic parameters in patients without and with AF recurrence.

	Before ablation	1 day	3 months	6 months	12 months
HR (beats/min)					
No rec	60.8 ± 9.3	75.3 ± 5.9*	64.3 ± 9.2	62.8 ± 8.6	64.2 ± 9.2
Rec	56.6 ± 5.4	77.5 ± 15.6*	66.5 ± 11.6*	72.2 ± 16.7*	64.9 ± 12.1*
LAV (cm^3)					
No rec	38.7 ± 11.3	40.7 ± 14.3	30.2 ± 9.8*	27.9 ± 8.1*	41.4 ± 8.0
Rec	48.4 ± 19.3	52.2 ± 17.5	39.6 ± 11.2	36.3 ± 7.5*	49.3 ± 16.4
LVDd (mm)					
No rec	48.5 ± 3.9	47.9 ± 3.6	46.8 ± 4.6*	46.7 ± 6.5	47.0 ± 3.1*
Rec	52.2 ± 6.5	49.2 ± 7.8*	48.9 ± 5.2*	48.9 ± 5.5*	50.4 ± 6.3
LVDs (mm)					
No rec	30.0 ± 3.9	28.3 ± 4.0*	29.0 ± 3.7	28.1 ± 4.5*	28.1 ± 2.7*
Rec	32.3 ± 8.4	29.1 ± 8.1*	30.9 ± 5.6	30.4 ± 6.0	32.7 ± 7.8
LVEF (%)					
No rec	67.9 ± 8.1	71.4 ± 6.3	68.2 ± 4.8	69.8 ± 7.3	70.5 ± 5.9
Rec	68.2 ± 11.3	71.7 ± 9.4	66.5 ± 8.6	66.7 ± 9.4	60.9 ± 13.6
E/A					
No rec	1.3 ± 0.5	1.3 ± 0.4	1.1 ± 0.3	1.2 ± 0.4	1.2 ± 0.3
Rec	1.0 ± 0.3	1.2 ± 0.4	1.1 ± 0.4	1.0 ± 0.2	1.2 ± 0.5
DcT (ms)					
No rec	179.5 ± 31.9	174.3 ± 29.0	202.9 ± 45.1*	188.8 ± 60.0	200.2 ± 40.9*
Rec	179.4 ± 33.4	185.6 ± 49.7	214.3 ± 45.2*	215.5 ± 48.2*	210.9 ± 65.1
E/e'					
No rec	6.5 ± 2.0	7.2 ± 2.3	6.2 ± 2.5	6.8 ± 2.5	6.6 ± 2.4
Rec	6.6 ± 1.8	7.9 ± 2.5	7.2 ± 3.0	6.8 ± 2.3	7.1 ± 2.0
Lateral a′ (ms)					
No rec	11.4 ± 4.2	13.6 ± 5.4	12.2 ± 2.9	11.8 ± 3.0	12.7 ± 3.3
Rec	13.6 ± 4.9	12.8 ± 4.3	10.9 ± 2.0	12.5 ± 4.9	10.3 ± 3.4
PA-TDI (ms)					
No rec	151.0 ± 14.6	148.9 ± 20.3	148.8 ± 22.7	146.8 ± 13.7	149.7 ± 19.1
Rec	163.9 ± 11.0	159.7 ± 18.4	166.0 ± 19.9	161.0 ± 20.9	156.9 ± 19.3

AF, atrial fibrillation; HR, heart rate; No rec, no recurrence of atrial fibrillation ($n=23$); rec, recurrence of atrial fibrillation ($n=10$); LA, left atrial volume; LVDd, left ventricular diastolic diameter; LVDs, LV systolic diameter; LVEF, LV ejection fraction; PA-TDI, time interval from onset of the P wave in ECG lead II to the peak a′ wave of the left lateral atrial wall; PA-TDI, time interval from onset of the P wave in ECG lead II to the peak a′ wave of the left lateral atrial wall. Values are the mean ± SD.

* $P<0.05$ vs. value before ablation.

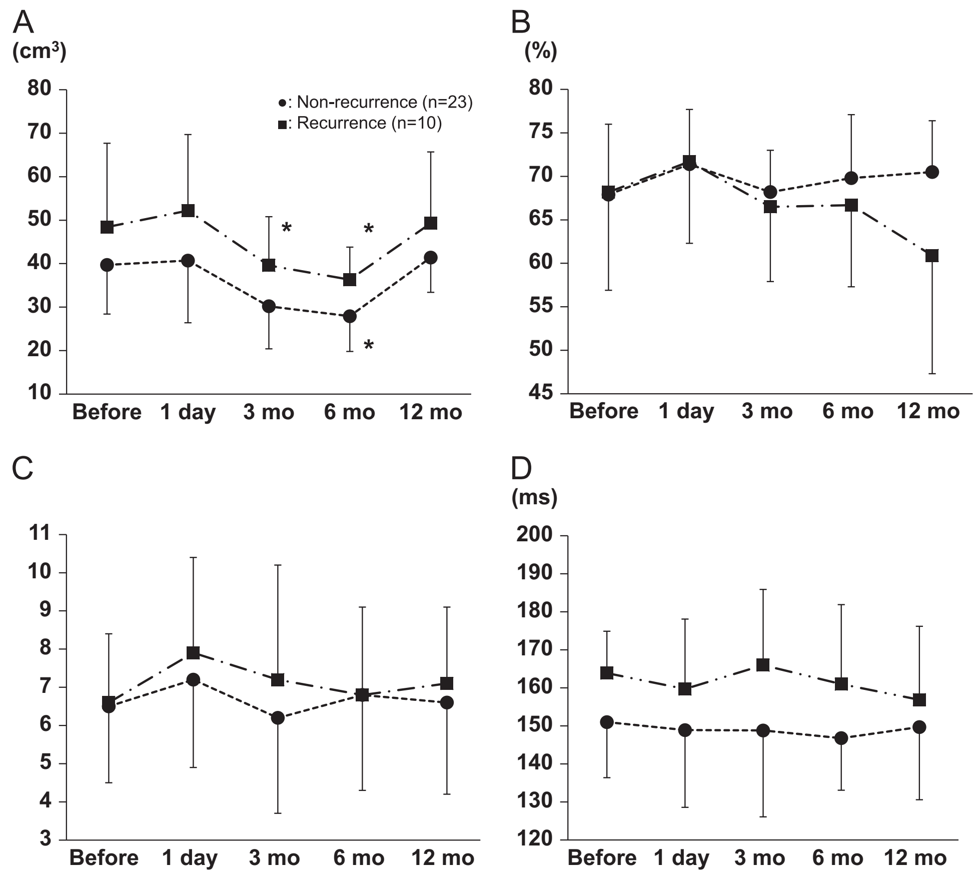

Fig. 2. Graphs showing transthoracic echocardiographic measurements in the patients without (●) and with AF recurrence (■) before and at 1 day, and 3, 6, and 12 months after the ablation. LAV, left atrial volume; LVEF, left ventricular ejection fraction; mo, months; PA-TDI, time interval from the onset of the P wave in ECG lead II to the peak a′ wave of the left lateral atrial wall. *$P<0.05$ vs. value before ablation. (a) LAV, (b) LVEF, (c) E/e′, and (d) PA-TDI.

LA-based ablation strategy on the PA-TDI interval, we compared the PA-TDI interval between baseline and day 1 after ablation in the patients who underwent PVI plus LA substrate-based ablation ($n=14$). The baseline PA-TDI interval was not prolonged at 1 day after PVI plus LA substrate-based ablation (159.8 ± 13.7 mm at baseline and 153.2 ± 21.3 mm after day 1, $P=0.1490$).

To exclude the effect of the use of class I anti-arrhythmic drugs, we assessed the PA-TDI interval in 22 (60.0% of the study patients) patients who were not prescribed class I antiarrhythmic drugs during follow-up. In this subgroup, there were no changes in the PA-TDI interval between baseline and 6 months (154.6 ± 16.4 ms vs. 149.9 ± 18.2 ms, $P=0.2001$) or 12 months after ablation (151.7 ± 20.5 ms, $P=0.4963$ vs. baseline value).

4. Discussion

The present study showed changes in specific echocardiographic variables during the 1-year follow-up period after AF ablation. The main findings were that (1) LAV was larger and PA-TDI interval was longer in patients with AF recurrence than in those without, (2) reverse LAV and LV diameter remodeling was noted, especially in patients with no AF recurrence, but there was no change in the PA-TDI interval, lateral a′, or LV systolic and diastolic function over 1 year regardless of any AF recurrence, and (3) there were no changes in the PA-TDI interval before and after ablation in patients with the extensive ablation strategy, even in those not using anti-arrhythmic drugs.

4.1. Relationship between baseline characteristics, general echocardiographic variables, and PA-TDI interval and AF recurrence post-ablation

In our patient series, AF recurrence was related to a relatively increased LAV and LV diastolic diameter. These variables have been shown to be hallmarks of progressive LA remodeling, as well as strong predictors of the recurrence of AF post-ablation [3–7]. Our patients who experienced AF recurrence also had a longer PA-TDI interval than patients without recurrence. Recently, the PA-TDI interval has been reported to correlate well with the signal-averaged *P*-wave duration [8], and that these variables can be used as non-invasively obtained predictors of TACT [6,8–10]. An increase in the TACT, which reflects the intra-atrial conduction time, can include slowing of atrial conduction and increased atrial dilatation, leading to a progression of LA remodeling [4,17,18]. LV diastolic dysfunction, valve incompetence, and an enlarged atrium, determined through echocardiography, are associated with a prolonged PA-TDI interval [19]. Therefore, a prolonged PA-TDI interval may be a surrogate marker for the existence of a substrate vulnerable to AF.

4.2. Changes in echocardiographic variables after AF ablation

We found that reverse remodeling of the LAV and LV diameters was observed by 6 months after ablation, especially in the non-recurrence group. These findings are consistent with previous reports that AF patients, in whom sinus rhythm was maintained after catheter ablation or cardioversion, had a significant reduction in LA size during the mid- and long-term follow-up periods [12–15].

Interestingly, we found that LAV and LV diameter returned to or below the baseline values at 12 months after ablation, despite the maintenance of sinus rhythm. Our findings suggest that reverse remodeling is a relatively short-term phenomenon that does not persist; i.e., some patients with non-recurrence of AF and a modest increase in LA dimension might experience a late AF recurrence. Although there is strong evidence that reverse remodeling in terms of LAV and LV systolic function occurs in the mid- to long-term follow-up period after ablation, whether reverse remodeling occurs in terms of LV diastolic function is controversial [12–15]. In the present study, the E/e' and E/A ratio did not change over 1 year in either the recurrence or non-recurrence group. Most recent studies have found no change or an increase in the E/e' ratio, despite improvement in the LA strain function, during the mid- to long-term follow-up period [12–14]. An earlier study highlighted the important contribution of AF suppression by RF ablation, especially for improving LV diastolic and systolic function [15]. The substantially higher baseline E/e' reported in that study might have allowed for a greater improvement than that seen in the present and other recently reported studies.

LVEF measured on day 1 after ablation did not change in either group. Over 1 year, it remained above the baseline level in the non-recurrence group, whereas it eventually decreased below the baseline level in the recurrence group. The small increase in LVEF at day 1 after ablation might be due to the change in the hemodynamic status that occurs after invasive ablation. However, the small increase remained for up to 6 months after ablation, indicating a link to the progressive reduction in LAV that occurred, especially in the non-recurrence group. This suggests that an improvement in LV systolic function by 6 months may be caused by the improvement in LA function.

4.3. Changes in the PA-TDI interval after AF ablation

Surprisingly, we found no change in the PA-TDI intervals during the follow-up period regardless of whether AF recurred. A majority of recent studies have shown that a significant decrease in the *P*-wave duration, signal-averaged *P*-wave duration, or TACT, measured on 3-dimensional maps, occurs in the acute phase and remains during the short-to-mid-term period after AF ablation [11,20–22]. These studies suggested that the shortening of the TACT that occurs after ablation might be caused by a conduction block in the PVs [11,20,21], reverse electrical remodeling [11], or a change in the breakthrough site in the LA, after the PV is isolated from the site of Bachmann's bundle to the medial septum [22]. The cause of an unchanged PA-TDI interval after AF ablation that we observed in our patient series remains unclear. One possibility is that LA substrate-based ablation might prolong the PA-TDI interval, which is overcome and thus masks the shortening of the TACT that results from a conduction block within the PVs or from reverse electrical remodeling. However, this speculated mechanism may not be the major cause; the baseline PA-TDI interval was not prolonged on day 1 after ablation in the patients who underwent PVI plus LA substrate-based ablation. The second possibility is that it was due to the effects of antiarrhythmic drugs used after ablation. Despite excluding data from patients using class I antiarrhythmic drugs, the PA-TDI interval did not change over 1 year. Thus, this effect appears to be minor. Rather, the phenomenon might occur from the different methodologies used to determine the PA-TDI interval compared with those used to determine *P*-wave duration or signal-averaged *P*-wave duration. The PA-TDI interval may overestimate the overall atrial activation time, resulting in no apparent effect by PVI, because the PA-TDI interval includes both the time required for the propagation of impulses from the sinus node area to the LA and the time required for the electromechanical coupling in the LA. For example, the offset of the PA-TDI was measured from the wall motion of the lateral LA very close to the mitral annulus; therefore, it may have been affected more significantly by the wall motion of mitral annulus in the LV end-diastolic phase rather than the exact wall motion of the lateral LA. This speculation might provide an answer as to why the PA-TDI interval was not affected by the ablation outcome, ablation strategy, or use of antiarrhythmic drugs.

4.4. Limitations

The present study had several limitations. First, the study included a small number of patients. Therefore, the number of patients was inadequate for application of a multivariate analysis to identify independent predictors of post-ablation AF recurrence. Second, we limited the echocardiographic measurements to conventional variables. An evaluation of LA strain or strain rate might provide more information, especially in regard to LA reverse remodeling [4,5,12–15]. However, we focused on LA reverse remodeling as evaluated with standard echocardiographic variables and the PA-TDI interval to increase applicability to clinical practice and to detailed time-based changes in these variables. Third, the correlation between the PA-TDI interval and TACT remains unclear because the TACT, determined by 3-dimensional geometric maps or (signal averaging) P wave duration, was not evaluated simultaneously with the PA-TDI. Finally, the incidence of AF was derived partly from symptom-driven ECGs and 24-h Holter recordings, which may have led to an underestimation of AF recurrence, because a large number of AF episodes are known to be asymptomatic.

5. Conclusions

In patients undergoing AF ablation, the PA-TDI interval might be a predictor of AF recurrence after ablation. Changes over time were observed in LAV and LV systolic and diastolic function, and these variables improved in patients without AF recurrence, but not in those with AF recurrence. Despite LA reverse remodeling, the PA-TDI interval was unchanged after more than 1 year of follow-up, regardless of the presence or absence of AF recurrence, possibly indicating that the wall motion of the lateral LA remains unchanged even after AF ablation.

Author contributions

Study concept/design: Yasuo Okumura.
Data analysis/interpretation: Hiroaki Mano, Yasuo Okumura.
Drafting the paper: Hiroaki Mano, Yasuo Okumura.
Critical revision of the paper: Hiroaki Mano, Yasuo Okumura, Ichro Watanabe.
Approval of the paper: Hiroaki Mano, Yasuo Okumura, Ichiro Watanabe, Masakatsu Ohta, Rikitake Kogawa, Naoko Sasaki, Toshiko Nakai, Kimie Ohkubo, Masayoshi Kofune, Koichi Nagashima, Kazumasa Sonoda, Hironori Haruta, Atsushi Hirayama.
Statistics: Hiroaki Mano, Yasuo Okumura.
Data collection; Masakatsu Ohta, Rikitake Kogawa, Naoko Sasaki, Toshiko Nakai, Kimie Ohkubo, Masayoshi Kofune, Koichi Nagashima, Kazumasa Sonoda, Hironori Haruta.

Disclosures and funding sources

None.

Conflicts of interest

None.

References

[1] Ouyang F, Bänsch D, Ernst S, et al. Complete isolation of left atrium surrounding the pulmonary veins: new insights from the double-Lasso technique in paroxysmal atrial fibrillation. Circulation 2004;110:2090–6.
[2] Lin YJ, Tai CT, Kao T, et al. Spatiotemporal organization of the left atrial substrate after circumferential pulmonary vein isolation of atrial fibrillation. Circ Arrhythm Electrophysiol 2009;2:233–41.
[3] Okumura Y, Watanabe I, Nakai T, et al. Impact of biomarkers of inflammation and extracellular matrix turnover on the outcome of atrial fibrillation ablation: importance of matrix metalloproteinase-2 as a predictor of atrial fibrillation recurrence. J Cardiovasc Electrophysiol 2011;22:987–93.
[4] Oakes RS, Badger TJ, Kholmovski EG, et al. Detection and quantification of left atrial structural remodeling with delayed-enhancement magnetic resonance imaging in patients with atrial fibrillation. Circulation 2009;119:1758–67.
[5] Tops LF, Delgado V, Bertini M, et al. Left atrial strain predicts reverse remodeling after catheter ablation for atrial fibrillation. J Am Coll Cardiol 2011;57:324–31.
[6] den Uijl DW, Gawrysiak M, Tops LF, et al. Prognostic value of total atrial conduction time estimated with tissue Doppler imaging to predict the recurrence of atrial fibrillation after radiofrequency catheter ablation. Europace 2011;13:1533–40.
[7] Zhuang J, Wang Y, Tang K, et al. Association between left atrial size and atrial fibrillation recurrence after single circumferential pulmonary vein isolation: a systematic review and meta-analysis of observational studies. Europace 2012;14:638–45.
[8] Merckx KL, De Vos CB, Palmans A, et al. Atrial activation time determined by transthoracic Doppler tissue imaging can be used as an estimate of the total duration of atrial electrical activation. J Am Soc Echocardiogr 2005;18:940–4.
[9] Chao TF, Sung SH, Wang KL, et al. Associations between the atrial electromechanical interval, atrial remodeling and outcome of catheter ablation in paroxysmal atrial fibrillation. Heart 2011;97:225–30.
[10] De Vos CB, Weijs B, Crijns HJ, et al. Atrial tissue Doppler imaging for prediction of new-onset atrial fibrillation. Heart 2009;95:835–40.
[11] Okumura Y, Watanabe I, Ohkubo K, et al. Prediction of the efficacy of pulmonary vein isolation for the treatment of atrial fibrillation by the signal-averaged *P*-wave duration. Pacing Clin Electrophysiol 2007;30:304–13.
[12] Kuppahally SS, Akoum N, Badger TJ, et al. Echocardiographic left atrial reverse remodeling after catheter ablation of atrial fibrillation is predicted by preablation delayed enhancement of left atrium by magnetic resonance imaging. Am Heart J 2010;160:877–84.
[13] Donal E, Ollivier R, Veillard D, et al. Left atrial function assessed by transthoracic echocardiography in patients treated by ablation for a lone paroxysmal atrial fibrillation. Eur J Echocardiogr 2010;11:845–52.
[14] Ollivier R, Donal E, Veillard D, et al. Early and late cardiac ventricular reverse remodeling after catheter ablation for lone paroxysmal atrial fibrillation. Ann Cardiol Angeiol (Paris) 2011;60:1–8.
[15] Reant P, Lafitte S, Jaïs P, et al. Reverse remodeling of the left cardiac chambers after catheter ablation after 1 year in a series of patients with isolated atrial fibrillation. Circulation 2005;112:2896–903.
[16] Ujino K, Barnes ME, Cha SS, et al. Two-dimensional echocardiographic methods for assessment of left atrial volume. Am J Cardiol 2006;98:1185–8.
[17] Spach MS, Boineau JP. Microfibrosis produces electrical load variations due to loss of side-to-side cell connections: a major mechanism of structural heart disease arrhythmias. Pacing Clin Electrophysiol 1997;20:397–413.
[18] Kostin S, Klein G, Szalay Z, et al. Structural correlate of atrial fibrillation in human patients. Cardiovasc Res 2002;54:361–79.
[19] Weijs B, de Vos CB, Tieleman RG, et al. Clinical and echocardiographic correlates of intra-atrial conduction delay. Europace 2011;13:1681–7.
[20] Ogawa M, Kumagai K, Vakulenko M, Yasuda T, et al. Reduction of *P*-wave duration and successful pulmonary vein isolation in patients with atrial fibrillation. J Cardiovasc Electrophysiol 2007;18:931–8.
[21] Udyavar AR, Huang SH, Chang SL, et al. Acute effect of circumferential pulmonary vein isolation on left atrial substrate. J Cardiovasc Electrophysiol 2009;20:715–22.
[22] Van Beeumen K, Houben R, Tavernier R, et al. Changes in *P*-wave area and *P*-wave duration after circumferential pulmonary vein isolation. Europace 2010;12:798–804.